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FILE 'HOME' ENTERED AT 16:52:47 ON 30 MAR 2000

=> file biosis

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FILE 'BIOSIS' ENTERED AT 16:53:26 ON 30 MAR 2000
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CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNS) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 29 March 2000 (20000329/ED)

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=> e reed,b/au

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E2	1	REED X B JR/AU
E3	0 -->	REED, B/AU
E4	2	REEDE D/AU
E5	1	REEDE D H/AU
E6	22	REEDE D L/AU
E7	1	REEDE DEBORAH/AU
E8	1	REEDE DEBORAH L/AU
E9	1	REEDE DEKKER T/AU
E10	1	REEDE J/AU
E11	1	REEDE J Y/AU
E12	1	REEDE JOAN/AU

=> e reed, berenice/au

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E9	1	REEDE DEKKER T/AU
E10	1	REEDE J/AU
E11	1	REEDE J Y/AU
E12	1	REEDE JOAN/AU

=> e gitomer, b/au

E1	16	GITOMER W L/AU
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E10	1	GITONGA K T K/AU
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E12	1	GITONGA N M/AU

=> s e2

L1 9 "GITOMER WILLIAM L"/AU

=> d l1 tot

L1 ANSWER 1 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
AN 2000:100698 BIOSIS
DN PREV200000100698

TI A comparison of fluoride bioavailability from a sustained-release NaF preparation (Neosten) and other fluoride preparations.
 AU **Gitomer, William L.** (1); Sakhaee, Khashayar; Pak, Charles Y. C.
 CS (1) Center for Mineral Metabolism and Clinical Research, University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX, 75235-8885 USA
 SO Journal of Clinical Pharmacology, (Feb., 2000) Vol. 40, No. 2, pp. 138-141.
 ISSN: 0091-2700.
 DT Article
 LA English
 SL English

L1 ANSWER 2 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 2000:18853 BIOSIS
 DN PREV200000018853
 TI Mapping a gene defect in absorptive hypercalciuria to chromosome 1q23.3-q24.
 AU Reed, Berenice Y. (1); Heller, Howard J.; **Gitomer, William L.**; Pak, Charles Y. C.
 CS (1) Center for Mineral Metabolism and Clinical Research, University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX, 75235-8885 USA
 SO Journal of Clinical Endocrinology & Metabolism, (Nov., 1999) Vol. 84, No. 11, pp. 3907-3913.
 ISSN: 0021-972X.
 DT Article
 LA English
 SL English

L1 ANSWER 3 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1998:496200 BIOSIS
 DN PREV199800496200
 TI Mutations in the genomic deoxyribonucleic acid for SLC3A1 in patients with cystinuria.
 AU **Gitomer, William L.** (1); Reed, Berenice Y.; Ruml, Lisa A.; Sakhaee, Khashayar; Pak, Charles Y. C.
 CS (1) Cent. Mineral Metabolism Clinical Res., 8885 Univ. Texas Southwestern Med. Center, 5323 Harry Hines Blvd., Dallas, TX 75235-8885 USA
 SO Journal of Clinical Endocrinology & Metabolism, (Oct., 1998) Vol. 83, No. 10, pp. 3688-3694.
 ISSN: 0021-972X.
 DT Article
 LA English

L1 ANSWER 4 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1998:93301 BIOSIS
 DN PREV199800093301
 TI 335-base deletion in the mRNA coding for a dibasic amino acid transporter-like protein (SLC3A1) isolated from a patient with cystinuria.
 AU **Gitomer, William L.** (1); Reed, Berenice Y.; Ruml, Lisa A.; Pak, Charles Y. C.
 CS (1) Cent. Gen. Mineral Metablism Clin. Res., Univ. Texas Southwestern Med. Cent. at Dallas, Dallas, TX 75235-8885 USA
 SO Human Mutation, (1998) Vol. 0, No. SUPPL. 1, pp. S69-S71.
 ISSN: 1059-7794.
 DT Article
 LA English

L1 ANSWER 5 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1997:29523 BIOSIS
 DN PREV19979932872
 TI Recent advances in the biochemical and molecular biological basis of cystinuria.
 AU Gitomer, William L.; Pak, Charles Y. C.
 CS Cent. Gen. Mineral Metabolism Clin. Res., Univ. Texas Southwestern Med. Cent. Dallas, Dallas, TX USA
 SO Journal of Urology, (1996) Vol. 156, No. 6, pp. 1907-1912.
 ISSN: 0022-5347.
 DT Journal; Article
 LA English

L1 ANSWER 6 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1995:479410 BIOSIS
 DN PREV199598493710
 TI In vivo effects of lipopolysaccharide on hepatic free-NAD(P)+-linked redox states and cytosolic phosphorylation potential in 48-hour-fasted rats.
 AU Gitomer, William L.; Miller, Bonnie C.; Cottam, G. Larry (1)
 CS (1) Dep. Biochem., Univ. Tex. Southwestern Med. Cent., 5323 Harry Hines Blvd., Dallas, TX 75235-9038 USA
 SO Metabolism Clinical and Experimental, (1995) Vol. 44, No. 9, pp. 1170-1174.
 ISSN: 0026-0495.
 DT Article
 LA English

L1 ANSWER 7 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1995:286547 BIOSIS
 DN PREV199598300847
 TI The in vivo effects of lipopolysaccharide on hepatic free NAD(P)+ linked redox states and cytosolic phosphorylation potential in 48 H fasted rats.
 AU Gitomer, William L.; Miller, Bonnie C.; Cottam, G. Larry
 CS Cent. Mineral Metabolism Clin. Res., Dep. Internal Med., Dallas, TX 75235-9038 USA
 SO FASEB Journal, (1995) Vol. 9, No. 6, pp. A1356.
 Meeting Info.: Annual Meeting of the American Society for Biochemistry and Molecular Biology San Francisco, California, USA May 21-25, 1995
 ISSN: 0892-6638.
 DT Conference
 LA English

L1 ANSWER 8 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1994:554344 BIOSIS
 DN PREV199598013892
 TI Metabolic hyperpolarization of liver by ethanol: The importance of Mg-2+ and H+ in determining impermeant intracellular anionic charge and energy of metabolic reactions.
 AU Veech, Richard L. (1); Gates, Denise N.; Crutchfield, Calvin; Gitomer, William L.; Kashiwaya, Yoshihiro; King, M. Todd; Wondergem, Robert
 CS (1) Lab. Metabolism Mol. Biol., Natl. Inst. Alcohol Abuse Alcoholism, 12501 Washington Ave., Rockville, MD 20852-1823 USA
 SO Alcoholism Clinical and Experimental Research, (1994) Vol. 18, No. 5, pp. 1040-1056.
 ISSN: 0145-6008.
 DT Article
 LA English

L1 ANSWER 9 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1993:137969 BIOSIS

DN PREV199395070769
 TI Determination of chloride potential in perfused rat hearts by nuclear magnetic resonance spectroscopy.
 AU Ramasamy, Ravichandran (1); Zhao, Piyu; Gitomer, William L.; Sherry, A. Dean; Malloy, Craig R.
 CS (1) Dep. Chem., Univ. Texas at Dallas, PO Box 850688, Richardson, Texas 75080
 SO American Journal of Physiology, (1992) Vol. 263, No. 6 PART 2, pp. H1958-H1962.
 ISSN: 0002-9513.
 DT Article
 LA English

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L1 9 "GITOMER WILLIAM L"/AU

=> d l1 ti abs ibib tot

L1 ANSWER 1 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
TI A comparison of fluoride bioavailability from a sustained-release NaF
preparation (Neosten) and other fluoride preparations.
AB Twelve normal subjects completed a crossover study with sustained-release
sodium fluoride (Neosten, 11.3 mg F), monofluorophosphate (MFP, 10 mg F),
and plain sodium fluoride (P-NaF, 11.3 mg F). After each preparation was
given with 400 mg calcium, serum fluoride (Fser) was measured for 24
hours, and pharmacokinetic data were calculated. Fluoride absorption in
the Neosten group, as measured by change in the area under the curve
(DELTAUAUC) of Fser, was less than 33% of that in the MFP and P-NaF
treated
groups. Both peak Fser (Cmax) and peak-basal variation in the Neosten
group were 25% that found in the other groups. t1/2 was nearly twofold
greater after Neosten. MFP and P-NaF showed greater bioavailability than
Neosten and much higher Cmax that exceeded the toxic threshold of Fser
(190 ng/ml). These findings could explain the ineffectiveness of MFP and
P-NaF observed in recent clinical trials.

ACCESSION NUMBER: 2000:100698 BIOSIS
DOCUMENT NUMBER: PREV200000100698
TITLE: A comparison of fluoride bioavailability from a
sustained-release NaF preparation (Neosten) and other
fluoride preparations.
AUTHOR(S): Gitomer, William L. (1); Sakhaee, Khashayar; Pak,
Charles Y. C.
CORPORATE SOURCE: (1) Center for Mineral Metabolism and Clinical Research,
University of Texas Southwestern Medical Center, 5323
Harry
Hines Boulevard, Dallas, TX, 75235-8885 USA
SOURCE: Journal of Clinical Pharmacology, (Feb., 2000) Vol. 40,
No.
2, pp. 138-141.
ISSN: 0091-2700.
DOCUMENT TYPE: Article

LANGUAGE: English

SUMMARY LANGUAGE: English

L1 ANSWER 2 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS

TI Mapping a gene defect in absorptive hypercalciuria to chromosome 1q23.3-q24.

AB Absorptive hypercalciuria (AH), a common cause of kidney stones, is due to

of intestinal hyperabsorption of calcium. The presence of a family history

of nephrolithiasis, in about half of the affected individuals studied indicates that an inherited genetic defect is one likely cause of AH. Although it is known that intestinal calcium absorption is regulated by a number of factors, the molecular biological basis for the increased calcium absorption in AH is unknown. This study was designed to determine the chromosomal locus of the gene defect linked to the AH phenotype in three families with a severe form of AH. Three kindreds were evaluated in a systematic autosomal genome-wide linkage analysis study. The AH phenotype, characterized by hyperabsorption of calcium and

hypercalciuria,

was linked to only one chromosomal locus, 1q23.3-q24. A 2-point logarithm of odds score of 3.3 was obtained with markers D1S318 and D1S196 at a recombination frequency of $\theta = 0$. Nonparametric multipoint linkage analysis yielded a peak nonparametric linkage Zall-score of 12.7, $P = 6 \times 10^{-6}$. Analysis of key recombinants within the families studied localized the gene to a 4.3-megabase region between markers D1S2681 (centromere)

and

D1S2815. A trait associated with intestinal hyperabsorption of calcium in a severe form of absorptive hypercalciuria has been mapped to chromosome 1q23.3-q24.

ACCESSION NUMBER: 2000:18853 BIOSIS

DOCUMENT NUMBER: PREV200000018853

TITLE: Mapping a gene defect in absorptive hypercalciuria to chromosome 1q23.3-q24.

AUTHOR(S): Reed, Berenice Y. (1); Heller, Howard J.; Gitomer, William L.; Pak, Charles Y. C.

CORPORATE SOURCE: (1) Center for Mineral Metabolism and Clinical Research, University of Texas Southwestern Medical Center, 5323

Harry

Hines Boulevard, Dallas, TX, 75235-8885 USA

SOURCE: Journal of Clinical Endocrinology & Metabolism, (Nov., 1999) Vol. 84, No. 11, pp. 3907-3913.

ISSN: 0021-972X.

DOCUMENT TYPE: Article

LANGUAGE: English

SUMMARY LANGUAGE: English

L1 ANSWER 3 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS

TI Mutations in the genomic deoxyribonucleic acid for SLC3A1 in patients with

cystinuria.

AB Cystinuria is an inherited transport disorder characterized by defective renal resorption of cystine and other dibasic amino acids. We have

studied

the occurrence of mutations in the SLC3A1 gene, which codes for a dibasic amino acid transporter-like protein, in 33 unrelated cystinurics. We

found

mutations in 34 of the 66 chromosomes studied. There were 14 different mutations in our study population, 8 of which had not been previously described. Of these new mutations, 4 were missense mutations: G1934C, C1259G, T1607G, and G1373A. The other 4 mutations consisted of a single base insertion mutation (2022 ins T), a single base deletion mutation

(163

del C), a 23-base deletion mutation (del 782A-804A), and a complex

mutation that consisted of a 36-base deletion (del C431-3 to T463) and a duplication insertion of 468 T to 474 A after nucleotide 474.

ACCESSION NUMBER: 8:496200 BIOSIS
DOCUMENT NUMBER: PREV199800496200
TITLE: Mutations in the genomic deoxyribonucleic acid for SLC3A1 in patients with cystinuria.
AUTHOR(S): Gitomer, William L. (1); Reed, Berenice Y.; Ruml, Lisa A.; Sakhaee, Khashayar; Pak, Charles Y. C.
CORPORATE SOURCE: (1) Cent. Mineral Metabolism Clinical Res., 8885 Univ. Texas Southwestern Med. Center, 5323 Harry Hines Blvd., Dallas, TX 75235-8885 USA
SOURCE: Journal of Clinical Endocrinology & Metabolism, (Oct., 1998) Vol. 83, No. 10, pp. 3688-3694.
ISSN: 0021-972X.
DOCUMENT TYPE: Article
LANGUAGE: English

L1 ANSWER 4 OF 9 BIOSIS COPYRIGHT 2000 BIOSIS
TI 335-base deletion in the mRNA coding for a dibasic amino acid transporter-like protein (SLC3A1) isolated from a patient with cystinuria.
ACCESSION NUMBER: 1998:93301 BIOSIS
DOCUMENT NUMBER: PREV199800093301
TITLE: 335-base deletion in the mRNA coding for a dibasic amino acid transporter-like protein (SLC3A1) isolated from a